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VULVOVAGINITIS OF SWINE

BY

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INTRODUCTION

In 1928 McNutt, Purwin and Murray published, under the above title, a report on an interesting condition in swine which occurred extensively in Iowa, U.S.A., and adjoining states, during the summer of 1927. The animals affected were young sows, the majority of which ranged from piglets six weeks of age up to 150-pounders. Young suckling females were also involved, although not so seriously. The vulvae in affected sows became very much enlarged, tense and elevated. In approximately 30 per cent. of cases there was prolapse of the vagina and in 5 to 10 per cent. prolapse of the rectum. Slight enlargement of the mammary glands was noticed in some sows. Deaths from secondary conditions such as haemorrhage, septic cystitis and uraemic poisoning occurred only among those showing prolapses. McNutt and his co-workers experimentally reproduced the condition by feeding mouldy maize. The extensive outbreaks were attributed to damage to maize crop by early frost and unusually wet weather in the previous year. It was the wettest September on record in Iowa.

In 1937 Pullar and Lerew recorded a similar condition in Australia. Practically all the females on two farms were affected and the occurrence of the disease was again associated with the feeding of mouldy maize. No further references to this condition have been found in the literature available.

In view of the rarity of this affection it is the purpose of this article to record two outbreaks of a similar nature in Ireland.

DESCRIPTION OF OUTBREAKS

OUTBREAK I.—In September, 1951, Mr. J. J. Pomeroy, M.R.C.V.S., Millstreet, Co. Cork, reported to the laboratory that he had encountered an unusual condition in a herd of swine. His client had two lots of pigs consisting of :—

Lot A—30 store pigs, 12 to 14 weeks old.

„ B—One sow with litter of pigs five weeks old.

All the females in Lot A showed swelling of the vulva and one had a prolapse of the vagina. The sow and all the female piglets in Lot B also showed vulvar enlargements. The diet consisted of ground barley, oats and waste wheat. A sample of the barley was forwarded and proved to be of very poor quality. It had a musty odour and the majority of the grains were visibly damaged and dark in colour. Unsuccessful attempts were made to obtain a quantity of the barley for a feeding experiment.

OUTBREAK II.—On 21.2.52 Mr. T. A. Mernagh, M.R.C.V.S., Gorey, Co. Wexford, reported a similar condition. His client had a total of 22 pigs consisting of the following lots :—

Lot A—Litter of 12 pigs four months old (seven females and five castrated males).

„ B—One gilt six months old.

„ C—Eight brood sows.

„ D—One stock boar.

All seven females in Lot A and the one in Lot B were showing enlargement of the vulva and two in Lot A had prolapse of the vagina. None of the brood sows or the stock boar was affected.

On 22.2.52 the owner brought to the laboratory a female suffering from vaginal prolapse. A clinical examination of this pig made the same day gave the following data :—

Age four months ; weight 25·5 lb. ; bright ; vulva swollen and tense, overlying skin normal ; protruding vagina bright red.

Width of vulva at base, 37 mm.

Length of vulva, 17 mm.

Length of protruding vagina, 20 mm.

Development of mammary glands was apparent and took the form of circular weals around the teats, varying in diameter from 32 to 37 mm.

The owner stated that the litter of 12 pigs (Lot A) were weaned at eight weeks and were given the following ration :—

Ground oats	1 part
Ground barley	1½ parts
Bran	½ part
Vitamin mineral protein ...	8 per cent.

This ration was also fed to Lots C and D from October, 1951, and Lot B from the beginning of January, 1952. Swelling of the vulva was observed in Lot A about 14 days after weaning, *i.e.*, around 20.1.52, and in the young sow (Lot B) a few days earlier. The swelling gradually increased until prolapse of the vagina occurred, as previously stated, in two sows in Lot A.

The barley used in the ration was homegrown Ymer variety. The owner observed that the grain was darker than usual when threshed and owing to dampness, it was turned daily for three weeks.

The premises were visited by the author on 27.3.52, seven days after withdrawal of the barley from the ration. According to the owner a considerable improvement had taken place since then in all affected animals. Vulvar enlargements were still apparent in the six remaining females in Lot A and varied in width from 23 to 27 mm. The prolapse in one sow had been reduced by surgical intervention. The vaginal mucous membrane appeared normal in every case, without any trace of an exudate. The swollen labiae were pinkish and resembled the appearance at oestrus. Mammary development, similar to that observed in the sow examined in the laboratory, was present in four of the sows. The larger sow (Lot B) was similarly affected, the vulva measuring 37 mm. transversely. The mammary glands were not examined in this case. The five castrated males in Lot A showed no abnormalities. The stock boar and brood sows appeared normal and several of the latter were approaching full term.

A quantity of the mouldy barley was procured and brought to the laboratory for a feeding experiment.

EXPERIMENTAL REPRODUCTION

Materials and Method.—Two littermate females, 11 weeks old, were weighed, their vulvae measured and abdomens examined for mammary development. They were then placed together in a clean sty and bedded with wheat straw.

A quantity of the mouldy barley, which was rather dark

and musty, was ground and weighed amounts were mixed with water and fed according to appetite.

Results.—The results obtained are presented in the following table :—

Pig number	Age beginning of experiment (days)	Weight beginning of experiment (lb.)	Weight end of experiment (lb.)	Duration of experiment (days)	Barley consumed combined total (lb.)	Width of vulva, beginning of experiment (mm.)	Width of vulva, end of experiment (mm.)	Nett increase vulvar width (mm.)	Mammary development beginning of experiment	Mammary development end of experiment
B17	79	37·0	41·5	13	38	16	24	8	Nil	Nil
B18	79	26·5	26·25	13		14	21	7	Nil	Present

The pigs did not relish the barley and during the first two days ate very sparingly. The vulvae were slightly swollen on the fifth day and continued to increase in size until the 11th day. The amount of barley consumed when swellings commenced was approximately 10 lb. The vulvae at the end of the experiment were tense, elevated and rose-pink in colour. The vaginal mucous membranes appeared normal and there was no evidence of an exudate. Mammary development was noticeable in Pig B18, the weals varying in diameter from 28 to 31 mm. There were no distinct weals in Pig B17 though the teats appeared to be slightly more elevated than usual. After withdrawal of the barley and return to normal rations the symptoms gradually abated, being barely noticeable 14 days later.

Phytopathology

A sample of the barley was submitted to Professor R. McKay, D.Sc., Department of Plant Pathology, University College, Dublin. In the course of his report he stated :—

(*a*) At least 90 per cent. of the grain was dead.

(*b*) Numerous fungi were present among which were *Fusarium graminearum* (conidial stage of *Gibberella zeae*) 1 to 2 per cent. and a species of *Cephalothecium* approximately 18 per cent. The latter was the commonest fungus present. There were many unidentifiable moulds in very small amounts.

(*c*) The only toxic fungi likely to occur in barley are *Claviceps purpurea* (ergot) and *Gibberella zeae* (*G. saubinetii* (*Mont.*) *Sacc.*). *Claviceps purpurea* was absent from sample.

(*d*) It is generally considered that approximately 10 per cent. of the grain should contain *Gibberella zeae* before it becomes toxic for pigs.

Christensen and Kernkamp (1936) investigated the toxicity of blighted or scabby barley for swine following a severe epidemic in the corn belt area of U.S.A. in 1928. Digestive disturbances were apparent shortly after ingestion, the most prominent symptoms being retching and vomiting. *Fusarium graminearum* inoculated on ears of growing maize was shown to produce a similar syndrome. Vulvovaginitis was not reported.

Discussion

The toxic substance in mouldy barley produces symptoms similar to those observed by McNutt *et al.* (1928) and Pullar *et al.* (1937) in young sows fed mouldy maize. The Australian workers have suggested that the active principle, following its excretion by the kidneys, has an irritant effect on the mucosa of the vagina and vulva. This, however, does not explain the development of mammary tissue. The author considers that the substance is more likely to be of the nature of an oestrogen which is absorbed from the alimentary canal and operates systemically. Libido was not displayed in the above experiment though McNutt *et al.* state that it occurred in some of their experimental pigs. They add, however, that the pigs were of such an age that they might have come in heat had they not been on experiment.

The condition is not likely to have any untoward sequelae if it is recognised early and the cause removed immediately.

Summary

(1) Two outbreaks of vulvovaginitis in swine are described.

(2) The occurrences were associated with the feeding of mouldy barley.

(3) The disease was experimentally reproduced in two young sows by feeding mouldy barley from one of the outbreaks.

(4) The theory that the ultimate cause is an oestrogen is advanced.

Acknowledgments.—I am grateful to Mr. J. F. Timoney, M.R.C.V.S., Director of this Laboratory, for his help in this investigation.

I am indebted to Professor R. McKay for examination of the grain and the loan of references.

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COMMON NAMES OF BRITISH INSECT PESTS

"In 1944," states *Nature*, "the Conference of Advisory Entomologists and the Pests and Diseases Committee of the Association of Applied Biologists set up a joint sub-committee to prepare a list of common names of the insect and related pests of garden plants, domestic animals, stored products and timber as they occur in Britain. The first part, dealing with slugs and snails, eelworms, beetles, hymenopterous insects and flies, was published in 1947. The second part, dealing with lice, thrips, plant bugs, aphids, and scale insects, Lepidoptera, fleas, mites and ticks, has recently appeared. Both parts consist of alphabetical lists of the accepted British common names (often with the American equivalents) and the corresponding scientific names; and alphabetical lists of the scientific names (under genera) and the recommended common name. These lists will be of great value to all applied entomologists in Great Britain in enabling them to secure some degree of uniformity in the names of their pests. Nor all the names will have equally ready acceptance by everyone. It is interesting to find the editor admitting quite frankly that 'by the very nature of the International Rules governing Zoological Nomenclature the scientific names must constantly be subject to revision.' It is intended to publish revised lists from time to time. Copies of the list can be obtained from Miss Barbara M. Stokes, Entomology Department, Rothamsted Experimental Station, Harpenden, Herts (Part 1, 2s.; Part 2, 3s.)."

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Our Parish Church.—Ten guineas, from the British Veterinary Association 36, Gordon Square, W.C.1, is among the latest donations to the St. Pancras Church Restoration Fund, and the total now stands at over £5,100. Altogether £20,000 must be raised to repair the ravages caused by dry rot and wood worm. Cheques should be made payable to the St. Pancras Church Restoration Fund and sent to the Vicar (the Rev. William P. Baddeley), St. Pancras Vicarage, 12, Fitzroy Square, London, W.1.

BIRD MIGRATION AND FOOT-AND-MOUTH DISEASE*

BY

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Are migratory birds to blame for the introduction of foot-and-mouth disease into this country? If so, which birds are involved and what is the extent of their responsibility? In this article the writers examine the evidence at present available on the subject.

The series of outbreaks of foot-and-mouth disease in the autumn of 1951 in the eastern counties of England gave rise to considerable speculation on the method by which infection was introduced into the country. There is a widely held opinion among laymen and veterinarians alike that the invasion was airborne and, in fact, introduced by migratory birds. The bird transmission theory is not original; it has been the subject of intermittent consideration for about 30 years. It is a theory obviously incapable of positive proof under the ordinary conditions of stock husbandry, so that any conclusions as to its validity must be based on the sum of circumstantial evidence. The purpose of this article is to examine the merits of the evidence at our disposal.

Origins of Previous British Infections

It is first necessary to consider the various methods by which disease is thought to have been introduced into this country in the past and to what extent they have a bearing on present-day infections.

Importation of Live Animals.—The earliest agreed record of foot-and-mouth disease in this country is for 1839, when there was a very widespread epidemic. In those days and for some 50 years after, there was a constant interchange of livestock between Britain and the Continent which resulted in frequent waves of disease sweeping across this country until the Order of 1892 put an end to the trade. A considerable import trade from the Argentine came to an end by Order following the introduction of disease into that country in 1899. The North American trade which was extinguished on commercial grounds at the beginning of the First World War, and revived for a short period in the late 20s, has not since been resumed. We have, of course, during all that period, imported large numbers of stock from Ireland and continue to accept animals from there when available. This trade and occasional importations of pedigree stock from other countries continue, but only under conditions which would render the chance of introducing disease remote.

Importation of Animal Carcases.—When livestock ceased to be imported from the Continent, a large business in freshly killed meat developed. After various attempts at control, this trade was suppressed by the Importation of Carcases (Prohibition) Order of 1926, and the risk from this source was finally eliminated. With the development of refrigeration early in the present century, it became possible to import increasing quantities of chilled meat from other continents. The risk of introducing disease in this way was not appreciated until the work of the Foot-and-Mouth Disease Research Committee in 1926 established that the virus could survive for as long as two and a half months in chilled meat carcases. This resulted in the Boiling of Animal Foodstuffs and the Meat Wrapping Materials Orders, which have gone a considerable way towards controlling the dangers from imported meat. However, the fact that almost three-fifths of all initial outbreaks of disease in the last 12 years have been in swine, and almost invariably in swine fed on swill or having access to suspect material, indicates that the danger from this source is ever present and a factor to be continually borne in mind when enquiring into the origins of confirmed cases.

Animal Feeding-stuffs.—Early in the century imported feeding-stuffs were held to be responsible for occasional outbreaks. Concentrated foods, especially the various high protein cakes, are manufactured by methods which eliminate the possibility of virus survival. Hay and straw are imported in varying quantities according to season, but only from countries free from disease. It is considered that there is no longer any risk of infection being introduced in this way.

Packing Materials.—A large proportion of our imports of manufactured goods arrive packed in hay or straw, the further use of which is governed by the Packing Materials Order of 1925. Reports on outbreaks indicate the possibility of infection being conveyed by other materials as, for example, in the packing of Dutch bulbs and on fresh horticultural produce. These and other contacts with imported material are now regarded as minor risks.

Biological Products.—An isolated outbreak of disease in Gloucester in 1938 was proved to originate from treatment with an infected glandular product of foreign origin. The use of these preparations is now controlled by the Serum and Glandular Products Order of 1939.

Movements of Persons and Vehicles from Abroad.—There is no evidence of infection having been conveyed in this way and the possibility is thought to be remote.

Origins of Present-day Infections

From what has been said it seems likely that the only continuing major risk to our livestock is from imported animal carcases. Perusal of case histories in primary pig outbreaks shows, with almost monotonous regularity, reports of "pigs fed on swill," "pigs had access to foreign bones," "owner is a butcher," or variations of the same theme. With rare exceptions primary pig outbreaks occur in housed animals, and it is difficult to appreciate what other means there could be of introducing infection to stock kept under enclosed conditions. Reports on a number of primary cases in cattle also indicate the possibility of contact with swill or other infective material.

Our present Head Office case records date from the reorganisation of the Animal Health Division on April 1st, 1938, and they show that between that date and October 31st, 1950, there were 365 primary outbreaks of foot-and-mouth disease in Great Britain. Of these, 223, or three-fifths of the total, occurred in pigs, 134 in cattle and eight in sheep. For the purposes of further comparison, it is proposed to treat the primary cattle and sheep cases as one entity of 142. Careful study of case histories showed that the possibility of contact with swill or other imported material could not be excluded in 50 of these cases, leaving some 92 cases of undetermined origin. Further breakdown of the figures indicated a definite concentration of primary outbreaks in the eight coastal counties from Lincolnshire to Dorsetshire, with 49 of the 92 primary out-

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breaks of unknown origin occurring in this group. Details of these totals are given in Table I.

TABLE I

PRIMARY OUTBREAKS OF FOOT-AND-MOUTH DISEASE IN GREAT BRITAIN IN THE PERIOD 1.4.38 TO 31.12.50

	Cattle and sheep			Pigs
	Total primaries	Swill contact possible	Origin obscure	
Lincs to Dorset ...	58	9	49	32
Rest of Britain ...	84	41	43	191
Total ...	142	50	92	223

The eight coastal counties contain rather less than one-seventh of the total livestock of the country yet, as will be seen from Table I, the total number of outbreaks of obscure origin among cattle and sheep in this area was greater than for the remainder of the country. It cannot be maintained that the average concentration of population or industry or of livestock is substantially greater in these counties than in other areas of the country, or that any of the previously enumerated factors are especially applicable to the coastal areas. The influence of swill infection shows a close parallel to the numbers of livestock with 31 primary cases in the eight counties against 191 in the rest of the country, a proportion of 1:6.

There are thus substantial grounds for the contention that some factor other than those previously considered applies to the east and south-east coastal area. The obvious factor is proximity to the Continent and consequently a relatively greater exposure to invasion when the disease is active on the other side of the water barrier. The figures previously quoted cover a period deliberately chosen to avoid two periods of abnormal disease incidence in the coastal zone, the series of outbreaks beginning in October, 1937, and again in November, 1951. In 1937 a series of unconnected initial outbreaks began on October 16th, involving the six coastal counties from Lincolnshire to East Sussex. Disease subsequently spread to the south-western counties and 268 cases were confirmed up to March 9th, 1938, when the outbreak was finally controlled. In 1951, after the country had been clear of the disease for some three and a half months, a further series of unconnected initial outbreaks occurred in the eastern counties between November 14th and 18th. In the five days 19 cases of disease were confirmed between Scarborough on the North Riding-East Riding border and Writtle in Essex. Three single cases occurring in the West Riding, Nottinghamshire and Leicestershire did not spread, but the coastal infections continued to spread until 106 outbreaks were confirmed before the disease was finally brought under control.

AIRBORNE INFECTION

Our present knowledge of the pathogenesis of the disease does not permit any other conclusion than that these mass invasions were due to airborne infection. It has been suggested that the virus of foot-and-mouth disease, which is amongst the smallest of all known viruses, might be transmitted in particulate form by air currents alone, much in the fashion that pollen grains have been carried for very considerable distances. There are some grounds for this supposition. In field outbreaks in dry, windy weather fine strings of saliva from infected animals may be seen being carried off in the wind current. How far this material will travel and how long virus will remain viable under such conditions is a matter for conjecture, but where this has been observed further outbreaks of disease have been exceptional and limited to premises in the vicinity. There has been no suggestion at any time that the virus may be carried over land for as far as 22 miles, which would correspond to the shortest over-water route from the Continent, and we have no reason to believe that virus will travel further in the relatively cooler sea atmosphere than it will over land. So far as east coast infections are concerned, the distances involved—at least 100 miles—and the prevailing adverse wind currents completely rule out particulate transmission.

Thus we are reduced to birds as carriers. The two major epidemics in 1937 and 1951 coincided with waves of widespread infection on the Continent, particularly in Holland and Belgium. They also coincided with the seasonal migration of various species of birds from their summer haunts in the Baltic countries and Central Europe to winter quarters in Britain.

THE BIRD TRANSMISSION THEORY

The earliest reference to birds as vectors of infection seems to have been made by Mettam (1914) in a consideration of the possible origins of the Irish outbreak of 1912. He laid no special emphasis on birds in general but neither did he entirely rule them out of reckoning. Sir Stewart Stockman in his annual report for 1919 considered birds to be improbable carriers but thought the possibility of airborne transmission deserved further enquiry. In 1923 Stockman and Miss Garnett made an attempt to correlate bird migration between the Continent and Britain with the then unexplained primary outbreaks. They set out to establish a negative by endeavouring to eliminate rather than incriminate birds. No particular species was considered, the authors confining themselves to a general treatment of the subject and concluding "the circumstantial evidence as a whole is very far short of establishing a negative case." As, at that time, the rôle of the swill tub was not appreciated, conclusions of this nature were vitiated in advance. In 1942 Bullough published an extensive study on the relationship of starlings and foot-and-mouth disease. He gave an excellent résumé of the ornithological aspects of the case and then attempted to establish a prima facie case against the starlings on purely statistical grounds, both as an agent in the autumn infections of this country and spring infections of the Baltic countries. Surprisingly enough, for it was then 1942, he also failed to appreciate the rôle of the swill tub and, with a limited understanding of the factors involved in the spread of the disease, was unable to assess correctly the value and meaning of his not inconsiderable data. Following a critical review of Bullough's paper by Dr. Landsborough Thomson in 1943, there does not appear to have been any further attempt to deal with the subject other than in the form of topical articles in the Press.

The bird transmission theory was at one time the subject of some small-scale investigations by Eccles at Pirbright. There, great difficulty was experienced in clinically infecting wild birds. One starling was so affected but the disease did not spread to other birds kept in close contact. Again using starlings, it was found that birds infected by mouth excreted virus in their droppings from 10 to 26 hours after ingestion of contaminated material, but there was no suggestion that virus multiplied in the system of the bird. After external contamination of feet and feathers, virus was

recovered up to a maximum of 91 hours. Birds exposed to natural infection and flown directly to healthy animals transmitted infection with one of three viruses. Birds similarly exposed to natural infection and then held in isolation for four to five hours failed to transmit the disease.

There is, of course, no possibility of reproducing these trials under completely natural conditions. It is highly probable that exposure to the diluting effect of air currents and rain results in some shortening of the viability of virus in external contaminations, but this could well be offset by the lower temperatures. Equally, removal of the fear complex in the natural state might reasonably be expected to prolong the period before complete elimination from the alimentary tract. For practical purposes we must assume that birds in their wild state do not become clinically affected with the disease or, in other words, do not become manufacturers of virus. But they may carry infection on their bodies for a time, probably measurable in hours, dependent upon the extent of the original contamination in relation to the prevailing weather conditions, and they definitely harbour infection for up to 26 hours after ingestion of contaminated material. Thus they are merely vehicles in the mechanical transmission of virus. On this basis, the rôle of any species of birds as potential vectors of foot-and-mouth disease can be assessed in mathematical terms as a function of their numbers, habits and opportunities for acquiring infection.

Birds have been blamed for the local spread of infection from known foci of disease in this country. Outbreaks in grazing stock, especially where outside trough feeding is practised, provide excellent conditions for external contamination of birds and for the ingestion of infective material. Reports from field staff on outbreaks in trough-fed sheep in downland country frequently comment on the large numbers of birds observed congregating around or on the flocks. In such circumstances the time involved in moving from one flock to another is only a matter of minutes. One cannot escape the conclusion that in field infections the mobility of birds makes them highly probable and dangerous disseminators of infection.

Bird Migration

On the wider question of introducing disease from the Continent, it is necessary to study the movements and species of birds which could be incriminated.

Our British birds may be divided into four groups:—

1. Native species which remain within the confines of our shores at all seasons.
2. Native species which leave our shores for warmer climates each autumn and return in the spring.
3. Birds from the Arctic region, northern and central Europe and the Low Countries which winter with us to escape the rigours of winter in their breeding areas.
4. Birds as in group 3 which merely use this country as a port of call on their way to warmer climates.

The general direction of the autumn migration of species which affect Britain is shown in Fig. 1. In the main, the spring migration follows the autumn lines in reverse.

Fundamentally, the autumn movements of birds are the sequel to falling temperatures reducing the available food supply in their breeding areas, but throughout countless ages these flights have become such an integral part of the lives of the birds that they are now performed almost instinctively. It may be that the movement also serves a useful purpose as an application of nature's law of "the survival of the fittest," in that immature or weakly birds unable to make the journey would inevitably perish during the winter in the breeding areas.

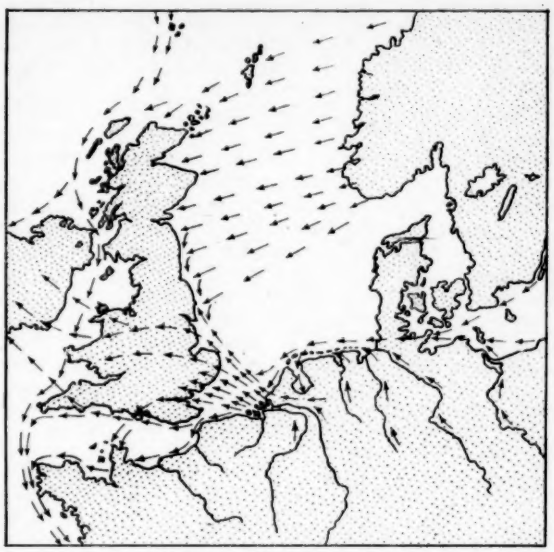

Fig. 1.—Map showing the principal migration routes of birds to the British Isles.

Our autumn visitors come from two distinct sources conveniently considered as northern and east-west. The northern stream, originating from Iceland, Norway and Arctic Europe, strikes our shores in the Shetlands, Hebrides and from Cape Wrath to the Humber Estuary. Some portion of the stream remains to winter in coastal or inland areas, but the majority skirt our coasts in a general southerly direction on their way to more temperate climates.

The east-west stream is drawn from the Baltic area, Central Europe and the Low Countries. Migrants from Central Europe travel down the river valleys of Germany to join birds from the Baltic and then skirt the coastline in a south-westerly direction to the Low Countries, where they may congregate in great numbers before making the sea crossing. Given favourable weather conditions, groups of birds may make a direct crossing to England from the Friesian Islands, and even from Denmark, but the majority follow the coastline to the estuaries of the rivers Rhine, Maas and Scheldt, thus ensuring the comparatively short sea crossing.

Except when made as a sudden sequel to sudden changes of weather, autumn migration is a fairly leisurely process. Daily journeys over land may amount to no more than 50 miles, so that the major part of each day is available for foraging. Sea crossings are made at a speed of 30 to 40 miles per hour. Thus birds may travel from Holland to Norfolk in three to four hours or to Lincolnshire in six hours.

Flight over water is generally directed due west, and the greater mass of the birds strikes our shores between Norfolk and the Thames estuary, with small numbers making landfall as far north as Lincolnshire. The majority penetrate inland and remain with us throughout the winter. Some, which have gone inland in the region of the Thames estuary, make a short overland journey to join with a stream skirting the Kent-Sussex coast and either continue on to winter quarters in Ireland or cross back to France. Weather conditions have a definite bearing on migration. Unusually mild or cold spells may delay or hasten the departure of birds from their breeding areas. High winds

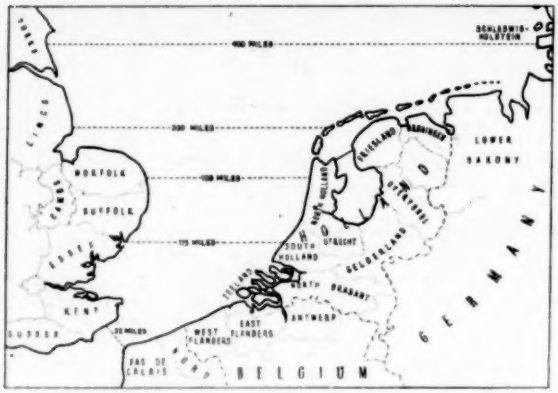

FIG. 2.—Distances travelled by migrating birds using the east-west route, showing also the relationship of coastal areas.

and rainstorms may hold up the sea crossing, so that large hordes of birds congregate in the normal areas of take-off, ultimately arriving in spectacular waves or "rushes." Adverse winds may tire migrants to such a degree as to affect the depth of their initial penetration into the country. In the same way, cross winds may blow birds off their normal course, with consequent landfalls at unusual points. In average years migration commences towards the end of September, rises to a maximum in the third week of October and is practically finished by the third week in November.

Later migrations on a much more reduced scale may take place at any time during the winter months. These are not really migrations at all but rather hard weather movements of birds which would normally winter on the Continent, being forced to seek warmer quarters by extremes of cold or snow.

There is nothing in previous records to suggest that the spring migration of birds has been responsible for introducing infection into Britain. No doubt several factors contribute to this position. Continental epidemics of foot-and-mouth disease tend to peter out during the winter months; housing of cattle on both sides of the Channel lessens the possibility of infection being picked up; birds have to make a longer sea crossing than their autumn counterparts; and last and most important, the spring movements of birds, being the result of a physiological urge, are performed in small groups and much more rapidly than the relatively leisurely autumn journeys where hordes of birds dally and feed frequently on the way.

Likewise there are no grounds for suggesting that disease has been introduced in the autumn by birds of the northern stream. Foot-and-mouth disease has rarely been confirmed in their areas of origin and certainly never in epidemic form. The majority of northern birds which winter with us do so in Scotland and Ireland, where primary outbreaks of undetermined origin are rare.

Bird Species in the East-West Migration

Having reviewed bird migration in general, and eliminated two of the three movements from further consideration, it is now necessary to study those species in the east-west stream which, on account of their habits and numbers, might be responsible for the autumn introductions of the disease into this country. A large proportion of our autumn visitors arrive as immense mixed flocks of rooks, jackdaws, starlings and skylarks. These four species are all essentially pasture feeders and thus have continuous opportunities for contact with grazing stock. As potential disease carriers, the differences between them are only a matter of degree based on their habits and numbers. Skylarks appear to avoid animals, rooks and jackaws do not avoid but rather ignore them, whilst the starling appears to make a special point of keeping close to grazing stock.

Continental lapwings arrive with the mixed autumn "rushes," making landfall from Yorkshire to Kent to winter very largely in the Humber and Thames estuaries. This species, in company with skylarks, also takes part in the winter hard weather movements from the Continent.

Wood-pigeons use the east-west route but are of less importance than most species, in that they do not regularly frequent pasture-land.

Wild ducks, chiefly mallard and teal, come to this country from the far north; many winter on the Continent. The continental birds, however, may be forced by severe weather to seek shelter on the mud flats and marshes of the south-east coast, where contact with cattle by way of drinking pools might readily be made.

Wild geese, particularly grey geese, migrate similarly to ducks and they also adopt winter migration when the marshes of the Baltic and Dutch coasts are frozen. Grey geese feed regularly on pastures and do not penetrate far inland.

Gulls of various species are common residents in this country. Following their breeding season, they migrate to the coast and with the onset of colder weather tend to move southwards, where they are joined on the south-east coast by continental visitors. Stormy weather at sea drives the gulls inland to roost in sheltered bays, inland lakes and secluded fields and marshes. The number of birds in a roost may be enormous, as in the case of Littleton Reservoir in Middlesex when the largest estimated total in 1940 was 45,000. Larger species of gulls are carnivorous and are great scavengers. Rubbish dumps, especially those containing waste food products and possibly meat and bones, are commonly visited by swarms of gulls, while ships in harbour or in coastal waters are besieged by gulls waiting to devour any waste foodstuffs. Undigested food is voided in the form of pellets, commonly found near the roosts and in fields and marshes. From their frequent and indeterminate wanderings in coastal areas, their propensity to feed on garbage and ship's swill, and their ability to regurgitate undigested food, it would seem fair to assume that gulls might readily constitute a direct vehicle in the transmission of disease to cattle and sheep from infected swill.

The Starling

The association of the starling with grazing stock and their pastures, which has already been noted, is a matter of common knowledge to anyone with practical experience of livestock husbandry. Starlings are almost omnivorous, but under normal conditions live largely on insects and molluscs. They tend to keep closely together in flocks which move over the pastures excavating for food particularly in the immediate vicinity of grazing animals and frequently within inches of their noses, almost as if the process of grazing was exposing suitable food material for them. It is not an uncommon sight to see starlings perched on the backs of cattle and sheep. In winter, when the ground is frozen or snow covered, starlings flock in the vicinity of houses and farms and groups of them enter cattle yards in search of grain or other edible material. No other British bird, whether native or migrant, shows such an affinity for close contact with livestock as does the

starling. When ground is contaminated with infective saliva the chances of picking up infection or otherwise becoming contaminated need no elaboration. Starlings have a further feature distinguishing them from most other species which may have a bearing on their disease-spreading propensities. During the hours of darkness migrant starlings roost communally in tall buildings in cities, in woods, in hedges and in reed beds which soon become heavily contaminated with their droppings. During the day they sally forth to feed in numerous small flocks, returning to the same point each evening. In roosts, which may contain up to 50,000 starlings, the birds are huddled so closely together that infection could readily be transferred from bird to bird. As to numbers, the starling is the greatest single factor in the east-west stream. Moreover, its numbers are thought to have increased substantially during this century and to be still increasing.

Summarising the foregoing on the basis that birds are mechanical vehicles in the transmission of virus, the starling by its habits and numbers is an obvious choice as chief suspect. This, of course, does not imply that other species can be entirely ruled out, but merely that their rôle is likely to be incidental to any played by the starling.

Comparison of British and Continental Infections In General

Applied to birds on migration, the habits and numbers of any of the species previously considered remain a fairly constant factor from year to year, the only variable being the degree of exposure to infection. Thus if birds are to be incriminated for introducing infection, we should expect to find a definite correlation between the extent of infection in certain areas of the Continent and the number of primary cases along our east and south-east seaboard. Looking over the records of British and continental infections since 1890, it is at once obvious that, apart from the years 1922-24 when the disease almost got out of control in this country, fluctuations in the incidence of disease in Holland, Belgium and the Pas de Calais area of France show a remarkable degree of similarity to the position in Britain. This is just as might reasonably be expected as, until the Importation of Carcases (Prohibition) Order came into operation in 1926, we were regularly importing potentially infected carcases from these countries.

Since 1926, enquiries into the origin of all outbreaks have been conditioned by the knowledge that infection may have been introduced through the agency of chilled meat products from abroad. We have been assisted in this by our ability to classify viruses by typing. Since 1930, with the exception of a short period in the early part of the Second World War, material has been submitted for typing from every outbreak not obviously connected with previous centres of disease, and has served a very useful purpose in correlating or separating the origins of parallel infections. This was particularly well demonstrated in the 1951 series of outbreaks when one geographically isolated case in Berkshire was typed as group "O," whilst material from some 20 parallel cases in the eastern counties were all group "A."

To determine the origin and status of any outbreak, it is essential to have individual case records. As available records date from 1937, further comparison will be limited to the period since that date. Table II gives comparative figures of total outbreaks for the months of September, October and November in each year since 1937 for Holland, Belgium and the Pas de Calais area of France and also the number of primary outbreaks in the south-east coastal counties of Britain.

Comparable figures for Denmark and Germany are not detailed, as they appear to have little or no bearing on British infections. For example, Denmark had only two outbreaks in the period September to November, 1937, when we experienced a major invasion. In the same months of 1938 Denmark had 69,883 outbreaks, whilst we had only three cases in the eastern counties. Again, in 1951, Western Germany, and especially the coastal provinces of Schleswig-Holstein and Hanover, experienced a very severe epidemic during the months of September and October, yet Britain remained free.

Table II indicates that there is a definite correlation between the disease position on the opposite shores of the shorter crossings of the North Sea. In seasons of high continental incidence our figures move up in sympathy, and conversely we may escape entirely when overseas

Table II

	Total number of outbreaks									Primary outbreaks in 8 coastal counties of Britain
	Holland			Belgium			Pas de Calais			
Year	September	October	November	September	October	November	September	October	November	
1937	11,444	37,998	31,230	4,090	14,098	22,321		(Epidemic)		20+
1938	34,905	39,623	17,257	3,642	3,816	4,960	191	37	68	7
1939	17,925	13,198	3,818	775	1,199	2,333	801	214	81	3
1940	66	53	34	1,758	1,027	547		(Occupation)		1
1941	2,492	2,191	904	141	105	67	125	93	59	—
1942	301	309	1,917	9	9	7	3	1	5	—
1943	9,708	7,618	2,605	—	2	6	1	—	1	1
1944	3,933	6,183	4,085	1,132	1,411	967	—	1	—	—
1945	28	38	73	2	2	23	—	—	—	—
1946	4,596	4,587	3,183	1,076	2,136	3,009	2,451	1,947	1,184	7+1*
1947	8	3	2	3	2	2	—	2	2	—
1948	17	48	339	448	773	272	406	563	458	3+1†
1949	200	245	324	14	6	3	50	14	19	2
1950	102	148	170	2	—	34	—	5	2	—
1951	109	3,485	12,357	47	6,261	23,613	15	27	196	20+

* Includes one case in Herts. † Includes one case in Wilts.

Note.—We have been warned that the continental figures for 1940–45, *i.e.*, the occupation years, are possibly exaggerated, as a report of foot-and-mouth disease was the invariable excuse given to the occupying authorities when inquiring into reduced farm output.

incidence is low. The monthly totals given are those for the whole of Belgium and Holland. However, the regular disease bulletins issued by their Governments detail the outbreaks of disease in the various sub-divisions of provinces of each country. Comparison of the dates of our primary south-east coast infections with such of these records as are available reveals that the danger to Britain is greatest and in fact only exists when disease has attained definite proportions in certain coastal provinces: in Belgium, the provinces of East and West Flanders; in Holland, the provinces of South Holland, North Brabant and, to a less degree, Sealand. These are the areas where the major part of our autumn visitors congregate before starting the sea crossing to our shores.

In 1937 and 1951 figures suggest that the risk is greatest on a waxing infection, which is in accordance with the accepted tenet that the infectivity of a virus is greatest in the earlier stages of an epidemic. That the coastal provinces of Belgium and Holland and the Pas de Calais zone of France are statistically the areas of menace to this country might be said to be a normal expectation. It does, however, restrict the potential danger zones to a much smaller area of the Continent than had previously been believed.

In Recent Years

The contention that the risk of introduction of disease by migratory birds is determined by the degree of infection in the areas of take-off for their sea crossing gains a large measure of support from the following table:—

Table III
Total Outbreaks of Foot-and-Mouth Disease in Belgium and Holland

	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.
1937	12	711	15,534	52,096	53,561	37,829	12,011
1951	32	31	156	11,459	35,920	22,802	—

In the 1937 series, disease first appeared on October 16th in Norfolk, with further primary strikes in that county, Lincolnshire, Suffolk and Essex before the end of the month. But in 1951 disease did not occur until November 14th, that is, 29 days later than in 1937. Examination of meteorological records fails to show any reason why migration in both years should not have been a continuous and unchecked process throughout the normal period. The autumn of 1951 was unusually mild, which may have resulted in some birds coming over later than usual and maybe after the end of the normally accepted migration period, but migration is such an ingrained instinct in birds that in the absence of adverse flying conditions it appears reasonable to assume that a comparable number of birds would have made the sea crossing by the same dates in each year and that the later appearance of disease in Britain in 1951 was entirely due to the later date of the build-up of disease in the Low Countries.

In dealing with an infection so highly contagious as foot-and-mouth disease, it is a matter of some difficulty to arrive at a decision as to which are primary or secondary outbreaks in invasions of this nature. It is accepted that as soon as symptoms of disease appear in any animal in these islands the risk of lateral spread is immediate. Making due allowance for this and other possible factors in the dissemination of virus, the primary outbreaks in the early stages of both invasions have been plotted in Fig. 3.

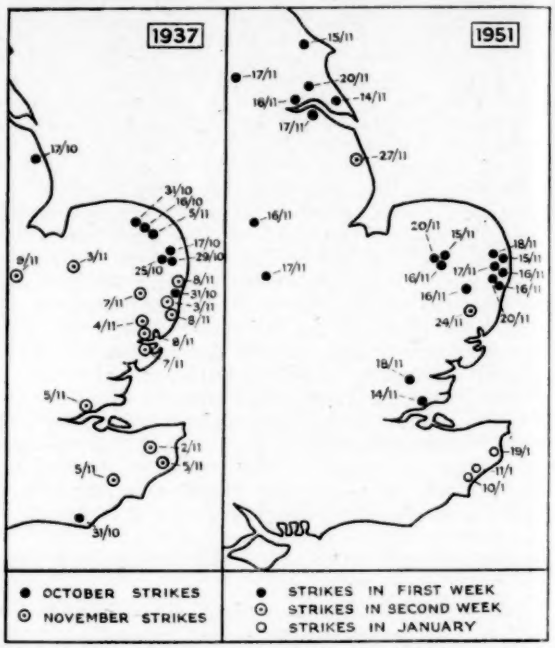

Fig. 3.—Location of primary outbreaks ("strikes") of foot-and-mouth disease in England in the early stages of the invasions of 1937 and 1951.

These indicate that in 1937 23 primary outbreaks occurred in the period October 16th to November 9th, involving all the coastal counties between Lincolnshire and East Sussex. Assuming the time interval from infection to confirmation of disease to be six days, then virus was being introduced into this country between October 10th and November 3rd, which coincides with the period of maximum extension of disease in the Low Countries in that year and also with the normal peak period of migration. All materials sent for typing were of a common strain "O" and identical with the type then decimating continental herds. The 1951 series started in a more explosive manner with 20 primary cases in the seven days November 14th to 20th. No cases occurred south of the Thames estuary, but there were no fewer than five north of the River Humber, an area not generally regarded as a major landfall for birds taking the east to west migration route. This may be explained by the constant south-west winds which prevailed during most of the autumn diverting birds northwards off their route with resultant landfalls further up the east coast. Using the same yardstick of six days from infection to confirmation of disease, the major crossings of the North Sea necessary to give rise to the 1951 infections would take place between November 8th and 14th, a period accepted by ornithologists as within the normal migration range. With the exception of the Berkshire case previously noted, all materials sent for typing were returned as strain "A," again identical with the type current on the Continent.

There can be little doubt that in both years further primary strikes occurred at dates later than those shown in Fig. 3, but secondary cases arising from original centres of disease progressively confuse the general picture until a point is reached where one is not justified in attempting to establish primary strikes, except in very isolated cases. In the 1951 diagram only two such cases are indicated, but

undoubtedly there were others. Reports from the field staff in both years stressed the large number of birds seen on infected premises or which had recently been noted on these premises. In nearly every instance the emphasis was on starlings.

As a continuation of the 1951 series, three primary outbreaks were confirmed in Kent between January 10th and 19th, 1952. These, the first cases south of the Thames, are of particular interest since they coincided with a definite extension of the continental epidemic into the Pas de Calais area of France. We have already made passing reference to the close parallel between British primary infections and the disease position in the Pas de Calais. During the five years 1941-45 the Pas de Calais was virtually free from infection. In that period the only primary autumn case in the south-east coastal counties was in Norfolk in 1943, at a time when Holland was experiencing a moderate epidemic. In 1946, however, when a wave of infection in the Low Countries was especially concentrated in the Pas de Calais, eight primary cases in grazing cattle were confirmed in South-East England as follow: October 18th, Dorset; November 9th, Suffolk; November 23rd, Suffolk; November 24th, Kent; December 4th, East Sussex; December 11th, Hertford; December 19th, Dorset; December 12th, Dorset. Material from every case was typed as " O "—the same type as prevailed across the Channel.

The sequence of the 1948 outbreaks was: November 4th, Wilts; November 23rd, Hants; November 23, Norfolk; December 12th, West Sussex. Again, all were in cattle on open grazing and again, all were typed to strain " O," as was the parallel French infection. It will be noted that south coast infections have tended to occur rather later in the year than those in East Anglia. The January, 1952, series in Kent follow the same trend. However, Fig. 3, with its comparison of 1937 and 1951 primary cases, shows four strikes south of the Thames between October 31st and November 5th, 1937. Although, unfortunately, we have not detailed French disease figures for 1937, the spread of the epidemic in that year was from France into the Low Countries, and at the relevant period the Pas de Calais was experiencing a major epidemic. Conversely, in the autumn of 1951 the disease spread relatively slowly from the Low Countries into France as the following figures show:—

TABLE IV

NUMBER OF OUTBREAKS OF FOOT-AND-MOUTH DISEASE IN PAS DE CALAIS 1951–52

Period to	15/9	30/9	15/10	31/10	15/11	30/11	15/12	31/12	15/1
Outbreaks	—	4	6	21	33	143	276	333	619

Again using the yardstick of six days from infection to confirmation of disease, the time of contraction of infection in the Kent outbreaks was between January 4th and 13th. The maximum expansion of disease across the Channel was in the period January 1st to 15th.

The sequence of events in the Channel area in December to January gives additional support to the previous contention that the prime essential for the spread of disease to this country is a certain level of infection in an appropriate area of the opposite shores. We appreciate that the period covered by this enquiry is not sufficient to justify any opinion being expressed on the apparent tendency for south coast outbreaks to occur later in the year than those in East Anglia. The impression may prove to be ill-founded but if correct may be due to later migrants making use of the shortest sea crossing in the Pas de Calais region or to hard weather movements of birds.

The Time Factor

Only one general point remains for consideration—the influence of the time factor. Experimental work by the Foot-and-Mouth Disease Research Committee established the capacity of the virus to remain viable under optimum conditions for as long as four months. Eccles's work at Pirbright gave optima of 91 hours for external and 26 hours for intestinal contaminations of birds. We have no means of assessing whether these times are maintained under natural conditions. Whether external or internal contamination of birds is the more probable means of carrying infection is a matter for future experiment. For the purposes of this article we accept both methods as possible.

Statistically, the danger areas are within five to six hours flying time of any of the normal landfall areas of our east coast. Migration from these areas is made in daytime and at a season when there are at least 10 hours of daylight. Even on the longest sea trip from Holland to Yorkshire, there is ample time for birds to feed both before and after the crossing and thus to have contact with animals in both countries in the same day. But the shorter the sea crossing the less will be the effect of rain and air currents in diluting surface contamination on a bird and of virus being eliminated from its alimentary tract, and consequently the greater will be the chance of viable infection being left on pastures, feeding troughs or on animals themselves.

That a time factor applies is obvious from the proximity to the coast of most initial strikes. It is not possible to say how far inland migrants penetrate before making a landfall. Doubtless this is largely determined by prevailing winds. But it is accepted that the majority of birds having made the sea crossing do not remain in coastal areas more than a day or two. The comparative absence of primary outbreaks in the Midland counties suggests that no great number of birds make a deep initial penetration of this country and also that they quite quickly become clear of infection. Applied to continental migration, this would explain why birds coming from or crossing the heavily contaminated region of Schleswig-Holstein and Hanover in September and October, 1951, did not bring disease to Britain; they " cleaned up " in Holland, probably leaving disease in their wake. Likewise, Low Countries birds in November, 1951, passing to the Pas de Calais would " clean up " in France before making the Channel crossing.

Conclusion

We have endeavoured to assess impartially the various factors having a bearing on the theory that birds on migration have been responsible for the introduction of foot-and-mouth disease into Britain. The seasonal incidence and location of primary outbreaks in the east and south coastal counties of this country shows a striking parallel to the period of autumn migration of birds and their area of landfall. We conclude that available evidence is adequate to establish a *prima facie* case against birds, and especially starlings on migration, as a means whereby foot-and-mouth disease is introduced into Britain; further that the frequency of its introduction is dependent on the extent of the disease in certain coastal areas of the Low Countries and north-east France at the season of bird migration.

NOTE.—Since this article was prepared there have been further outbreaks of foot-and-mouth disease in the spring

and summer of 1952. Many of these occurred in the southern coastal counties of England. At the same time there has been a build-up of infection in the northern coastal counties of France unprecedented in extent for the time of the year. The evidence suggests that in 1952 for the first time the spring migration may have been responsible for introducing infection into this country. Moreover, the continuance of further cases in the coastal counties throughout the summer while the intensity of infection in Northern France was still increasing raises the question whether in certain circumstances it is not possible for infection to be carried by cross-Channel movements of birds which are not part of the regular migratory movements.

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A VETERINARY COLLEGE FOR NEW ZEALAND?

In its issue of July 18th the *Wellington* (N.Z.) *Evening Post* reports that in the House of Representatives, on that day, a hint that the time might be considered ripe for the establishment of a veterinary college in New Zealand was given by the Minister of Agriculture (Mr. Holyoake), who added that a Committee of Investigation similar to that which sat is 1944 or 1945 would probably be set up to make recommendations to the Government. In that instance, a minority report had suggested that a college should be set up when there were 250 veterinary surgeon in New Zealand. With more than 200 now operating, that figure was being approached and the view of that minority might well be worth further exploration, said the Minister. He had no idea how much it would cost to establish a college, but a rough estimate of the cost of running it was between £30,000 and £40,000 a year.

The House was discussing the annual report of the Veterinary Services Council and some members urged that until a college was established in New Zealand more bursaries of greater value should be granted for New Zealanders to train in Australia, where about a dozen of them a year were joining for study.

* * * * *

Canada has banned the import of United States swine and pork products to prevent the introduction of vesicular exanthema. Dr. Childs, Veterinary Director-General, referring to the prevalence of this disease in about two dozen American States, said it was "a highly infectious disease of swine which if introduced into Canada could cause serious damage to the Canadian swine industry."

* * * * *

Weekly Wisdom

The improvement of man's estate by the application of scientific knowledge is one of the loftiest adventures; but a belief that it can be achieved by scientific methods alone, without a moral basis to society, is a perilous illusion.—Professor A. V. Hill, F.R.S. (Presidential Address to the British Association, 1952).

CLINICAL COMMUNICATION

DEATHS OF CATTLE FROM LEAD ARSENATE POISONING

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Introduction

This case history of a series of deaths of cattle eating lead arsenate is reported as being of some toxicological interest in that a wide range of dosage was given to all the animals in an entire herd. It is therefore possible to see the effect of dosages of various size on animals of different age, state of health and bodily condition.

History

The lead arsenate was fed instead of lime as an ingredient of the concentrate ration to a dairy herd at Grove Place Agricultural Station, Jamaica. The herd consisted at the time of 36 animals made up of 24 adult cows, nine young heifers and three young bulls. It was the practice to mix sufficient feed on each Thursday to last one week, and in the feed to mix a mineral mixture in the proportions set out below. On Thursday, December 27th, 1951, the feed was mixed as follows :—

1. Grain Feed

	lb.
Ground corn	1,000
Coconut meal	376
Rice bran	255
Total	1,631

to which was added

2. Mineral Mixture

	lb.
Common salt	40
Lime (CaO)	40
Dicalcium phosphate ...	20
Total	100

Of the 100 lb. mineral mixture made up on this date, 48 lb. was added to the 1,631 lb. grain feed. As transpired later, the lime was substituted by arsenate of lead kept for the spraying of potatoes.

At the 3 p.m. milking, the 36 animals were fed the amount of concentrate feed indicated in Table I.

Symptoms and Course

As far as can be ascertained, no immediate symptoms of illness were observed and no reliable information was obtained as to the quantities of grain feed (and mineral mixture) left behind in the feed troughs after the milking of cattle was finished and the cattle turned out to pasture. That night, nobody working in the dairy suspected that there was anything wrong with the feed or herd.

On the following morning, Friday, December 28th, 1951, at about 6.30 a.m., when the milk herd were being driven into the milk shed, it was observed that the cow "Rosita" was purging badly and that four or five other cows were also "scouring" and, according to the Livestock Officer, the animals' eyes had "a staring appearance, hair all over the bodies in an upright position, and they all looked droopy." Milk yield had dropped considerably and in some cases practically to nil. At the same time, it was noticed that the cows would not eat the grain feed in their troughs. By this

time most of the cows in the herd were showing symptoms of acute diarrhoea. The Livestock Officer then tasted the grain feed, found it "mouldy and slightly acid," and decided to check up on the ingredients used in the mixing on the previous day. It was then discovered that instead of 40 lb. lime (CaO), 40 lb. of arsenate of lead had been used for the mineral mixture.

The matter was reported to the author at approximately 9 a.m. The dairy was visited immediately and all the usual clinical symptoms of arsenical poisoning were to be seen: violent purging, tenesmus, staggering gait, great thirst, staring coat, etc. These were, however, confined to the 24 milking cows in the milking shed; the nine heifers and yearlings in another shed, except for slight diarrhoea in three or four cases, showed no symptoms of illness or discomfort, nor did the three young bulls that were turned out at pasture. Yet all of them—36 animals—were supposed to have had the grain feed plus the poisoned mineral mixture.

Examination showed that the following cows (1) T. F. Pearl, (2) Beryl, (3) M. A. Petunia, (4) Rosita, and (5) Standard Pioneer, were the most critical cases, with typical acute arsenical poisoning. Treatment of the whole 36 cattle was immediately undertaken, but T. F. Pearl and Beryl died that night, M. A. Petunia and Rosita on the 30th, and Standard Pioneer on the 31st. Another cow, R. D. Jewell, which showed great promise of recovery, relapsed and died on January 13th, 19 days after the poisoned feed was taken.

It should be noted that the lead arsenate mixture, according to the label, contained the equivalent of 62·64 per cent.

Name of animal	Age (months)	Condition	Concentrated food (lb.)	Lead Arsenate (calculated)		Yield of milk (1951)—lb.						Symptoms
						26.12	27.12	28.12	29.12	30.12	31.12	
				Arsenious oxide Dr.	Lead oxide Dr.							
Flythe	60	Good	5	2½	5	13·2	13·0	5·6	0·3	0·2	1·0	Purging
Torono Pearl ...	117	"	4	2	4	13·4	12·7	9·3	3·0	3·2	3·5	" but no tenesmus
" Honeybelle	102	"	4	2	4	9·2	6·7	2·9	0·3	0·2	0·3	"
" Pergola ...	129	"	4	2	4	13·2	12·8	3·4	2·6	2·5	4·4	" but no tenesmus or distress
T. G. Pergola ...	47	"	5	2½	5	13·1	13·0	4·4	0·2	0·7	2·0	Purging
P. D. " ...	72	"	7	3½	7	19·4	19·0	6·7	3·9	1·5	3·8	" but no tenesmus or distress
" Pearl	69	"	6	3	6	16·6	17·0	6·3	1·1	1·8	2·5	Purging
Nelsie	60	"	4	2	4	9·8	9·3	2·4	1·2	1·0	0·3	Violent purging; aborted January 2nd, 1952
O. A. Petunia ...	60	"	3	1½	3	4·2	3·8	1·2	In calf (drying out)			Violent purging; aborted January 10th, 1952
M. A. " ...	117	"	4	2	4	10·0	9·5	3·1	Nil	Died 30.12.51		Typical symptoms of arsenical poisoning
Roselle	53	"	5	2½	5	12·0	12·0	5·1	2·4	2·0	1·0	Purging
Setta	49	"	6	3	6	15·0	13·5	5·7	0·1	0·2	0·2	"
G. M. Flower ...	113	"	5	2½	5	12·2	12·2	6·3	2·1	0·2	2·5	"
T. F. Pearl	88	Poor	7	3½	7	15·2	18·2	1·2	Died 29.12.51			Typical symptoms of acute poisoning
Bridget 6th ...	80	Good	5	2½	5	10·9	12·0	1·3	1·0	1·1	0·9	Purging
R. P. Observer ...	74	"	8	4	8	9·0	8·0	2·1	1·7	0·6	0·2	" and distress
Owl's Perilla ...	140	"	10	5	10	22·6	20·5	5·1	Nil	Nil	4·5	Very acute; purging and great weakness; inability to stand for days; bottle fed and given stimulants and penicillin but gradually got weaker and died February 7th, 1952
Rosita	52	"	9	4½	9	24·0	22·0	2·9	Nil	Died 30.12.51		Typical symptoms of acute poisoning
Beryl	36	Poor	8	4	8	16·7	16·2	2·2	Died 29.12.51			Typical symptoms of acute poisoning
D. O. Mead ...	74	Good	9	4½	9	22·1	22·0	7·3	0·1	4·0	2·0	Purging
F. V. Honeybelle ...	41	"	10	5	10	21·7	18·7	10·9	5·6	4·0	6·5	Little if any symptoms
Standard Pioneer ...	122	"	8	4	8	18·0	15·4	4·7	1·2	Nil	Died 31.12.51	Typical symptoms of acute poisoning
R. D. Jewell ...	24	"	8	4	8	27·7	28·4	10·7	Nil	4·7	2·1	Typical symptoms; refused all food, was bottle fed, got gradually weaker and died January 13th, 1952
Tor. Golden Honey	26	"	8	4	8	Heifer heavy in calf						
H. O. T. Pioneer ...	14	"	4	2	4	Heifer						No clinical symptoms of arsenical poisoning—a few had "loose" motions which could be attributed to the saline given immediately after diagnosis
Concie	14	"	4	2	4	"						
H. O. T. Meade ...	15	"	4	2	4	"						
Minute	15	"	4	2	4	"						
H. O. V. Perilla ...	17	"	4	2	4	"						
Braid	18	"	4	2	4	"						
Ruby	18	"	4	2	4	"						
H. V. Pergola ...	9	"	4	2	4	"						
M. A. Pet. Chieftainess	10	"	4	2	4	"						
H. C. Perfect Chief	7	"	3	1½	3	Young bull						No ill effect
H. C. H'belle Chief	9	"	3	1½	3	"						
H. V. Pearl Chief...	6	"	3	1½	3	"						

lead oxide and 32 per cent. arsenic oxide. This being so, and providing that the grain feed and the poisoned mineral mixture were well mixed together, each pound fed would contain approximately 32 grains of arsenic and 62 grains of lead. As will be seen from the table, some cows received as much as 8, 9 or 10 lb. of feed and consequently a large amount of arsenic. At the same time, it is generally recognised that cattle are less susceptible than horses to the toxic action of arsenic and if in good condition will tolerate quite large amounts ; it is, therefore, very difficult in the case of cattle, to fix a toxic dose. Although it is generally stated that 4 to 8 drams will produce symptoms, as will be seen from the attached table, quite a number of the cows received 3, 4 and 5 drams.

At no time could symptoms of lead poisoning be recognised ; if present, they were obscured by the more acute arsenical symptoms, though one would have thought the yearlings and young bulls would have shown some cerebral symptoms, particularly as they showed no symptoms of arsenical poisoning.

Treatment

Immediately arsenical poisoning was recognised, and in view of the possibility of lead complications, a full dose o magnesium sulphate was given to every animal ; this was followed by daily dosing with sodium hyposulphite, either as a drench or by intravenous injection. This was carried on for some two to three weeks after the more severe symptoms had subsided. In the cases of M. A. Petunia, Rosita and Standard Pioneer, large doses of penicillin were given also.

Post-mortem Examination

Post-mortem examinations were carried out on Beryl and T. F. Pearl, and showed the usual intense gastro-enteritis and the typical rosy-pink condition under the skin.

Portions of the kidneys, liver, and stomach wall and samples of stomach contents, were sent for chemical examination and an extract from the report of the Government Chemist is given below :—

Analysis of Feed

Lead as Pb ... ... 0·47 per cent.
Arsenic as As_2O_3 ... 0·10 „

Analysis of Viscera

Beryl

A. *Kidney and Liver*
0·00014 per cent. As_2O_3 (0·005 grains arsenic in ca, 7¾ oz. submitted).
0·00021 per cent. Lead (0·008 grains in ca, 7¾ oz. submitted).

B. *Portions of Stomach Walls*
0·00016 per cent. As_2O_3 (0·004 grains arsenic in ca, 6 oz. submitted).
Lead not determined.

C. *Stomach Contents*
0·00046 per cent. As_2O_3 (0·003 grains arsenic in ca, 2 oz. submitted).
0·00025 per cent. Lead (0·002 grains lead in ca., 2 oz, submitted).

T. F. Pearl

A. *Kidney and Liver*
0·00016 per cent. As_2O_3 (0·007 grains arsenic in ca, 10 oz. submitted).

B. *Portion of Stomach Lining*
0·00017 per cent. As_2O_3 (0·002 grains arsenic in ca, 3 oz. submitted).

C. *Stomach Contents*
0·00021 per cent. As_2O_3 (0·005 grains arsenic in ca, 5 oz. submitted).
The lead was not determined for any of these three specimens.

Sequelae

Two cows, Nelsie and O. A. Petunia, aborted on January 2nd, 1952 and January 10th, 1952, respectively, 6 and 14 days after being fed the arsenical mixture.

On January 18th, following a suspected case of mastitis, the whole herd was subjected to the bromothymol blue mastitis test and almost every quarter showed a positive (blue-green) reaction. Two samples of milk, etc., were taken for examination, and were of normal *p*H (6·67 and 6·78). Quarter samples of milk from eight cows were examined bacteriologically for mastitis, *Streptococcus dysgalactiae*, *Streptococcus bovis* and *Streptococcus pyogenes* (animal type C), were each isolated from one cow. It appeared that the alkaline reaction must in some way have been caused by the after-effects of the arsenic.

Discussion

1. Several hours elapsed before symptoms of arsenical poisoning became apparent.

2. Of the five cows that died, on December 29th, 30th and 31st, 1951, T. F. Pearl and Beryl were the only two in " poor " condition, and they both died within 36 hours of partaking of the poisoned food ; the cows, M. A. Petunia and Standard Pioneer, were among the oldest cows in the herd, whilst Rosita was the first to be noticed with symptoms. The oldest cow in the herd—Owl's Perilla—made a gallant fight although showing acute symptoms and several times appeared likely to recover when the acute symptoms gradually subsided. Eventually, she died on February 7th, 1952, some six weeks after partaking of the arsenical food. The cow, R. D. Jewell, died on January 13th, 1952. This cow, like Owl's Perilla, appeared to be recovering but gradually got weaker and died, apparently as a result of the effect of the arsenic in the mucous membrane of the stomach and consequent inability to digest her food.

3. Two cows, Nelsie and O. A. Petunia, aborted on January 2nd and January 10th respectively, 6 and 14 days after consuming the arsenic.

4. The cow, T. G. Pergola, developed on January 17th what at first appeared to be mastitis but later almost the whole skin peeled off the udder and the four teats.

5. It is difficult to account for the fact that only the adult cattle showed symptoms of arsenical poisoning and that the nine heifers and three young bulls did not apparently show any ill-effects, despite the amounts of feed and consequently of arsenic (and lead) they received. It would appear that either the feed (and mineral mixture) were badly mixed or, more likely, they did not get any at all—it being the day after the Christmas holidays. The possibility of a correlation of the severity of symptoms with age cannot be ignored.

6. The apparent " alkaline reaction " of the milk of the whole herd when tested with bromothymol blue, on January 17th, could not be satisfactorily exlpained.

Director-General of Nature Conservancy.—The Nature Conservancy announce the appointment of Mr. E. M. Nicholson, the distinguished ornithologist, as Director-General on the retirement of the present Director-General, Captain C. R. P. Diver. Since 1945 Mr. Nicholson has been Secretary of the office of the Lord President of the Council. On the death of Mr. B. W. Tucker Mr. Nicholson became editor of the monthly magazine *British Birds*. In 1951 he published an extremely original volume on *Birds and Men*. Previous publications from his pen were *Birds in England* and *How Birds Live*. He was Chairman of the British Trust for Ornithology from 1947 to 1949.

ABSTRACTS

Massive Dose Penicillin and Streptomycin Treatment of *S. pyogenes* Mammary Infections*

The investigations recorded were carried out on a herd of some 200 cows which, during 1943, had been treated extensively for *Str. agalactiae* infection. Subsequent to the virtual elimination of this organism, mastitis associated with *S. pyogenes* began to assume appreciable proportions in the herd. In 1945, 52 per cent. of 226 cows were shedding staphylococci in their milk. By 1950, only 17, or 6 per cent., in the herd, which had reached 276 animals, were actually shedding *S. pyogenes*, this marked improvement being attributed to the measures taken. These consisted mainly of three different treatments apart from the routine hygienic control measures. Penicillin and streptomycin were used alone and in combination. A better effect was achieved by using the two together than separately and, moreover, the results indicated that treatment during lactation was somewhat more effective than during the dry period. Quarters were recorded as cured if they remained free from *S. pyogenes* for three months.

Twenty-two cases in dry cows were treated, using total doses of from 2,000,000 to 8,000,000 units of penicillin and 4 grammes of dihydrostreptomycin. (Most frequently the combination consisted of 1,000,000 units of penicillin and 1 gramme of dihydrostreptomycin in 50 ml. of sterile distilled water, infused four times at 24-hour intervals in lactating glands and at 24- or 48-hour intervals in dry glands.) Complete success was achieved in infections of less than six weeks' duration, and 80 per cent. in infections of from two to six months' standing, and 71 per cent. in cows infected 7 to 12 months. The rate of cure was, in fact, inversely proportional to the length of time that the infection had persisted. The argument put forward is that the longer the infection persists the more deeply do the organisms penetrate, until finally they reach the interstitial tissue, when they are much less susceptible to therapy than when existing superficially on the epithelium in the early stages of an infection.

N. S. B.

* **Effect of Massive Doses of Penicillin and Streptomycin on *Staph. pyogenes* Mammary Infections.** Schalm & Woods. (1952.) *Amer. J. vet. Res.* **13.** 26–30.

* * * * *

Origin of the Blood Fructose of the Foetal Sheep*

In pregnant ewes the only sugar in the circulating blood is glucose, but in the foetus the total reducing substances of the blood are glucose and fructose. In the ewe the blood glucose level is above that of the foetus, but in the foetus total reducing substance levels are substantially higher because of the fructose. Foetal blood fructose levels tend to be lowered towards the end of the gestation period.

Intravenous injection of glucose into the pregnant ewe produces the usual hyperglycaemia with return to normal levels in three to four hours. In the attached foetus the hyperglycaemia peak value lags by about 30 to 45 minutes, but returns to normal coincidentally with the dam. As the additional glucose reaches the foetus the foetal blood fructose level is elevated. When glucose is injected intravenously into the attached foetus a similar hyperfructosaemia is observed, but fructose never appears in the maternal blood.

The fructose is produced in the placenta and not in the foetus since fructose is formed following either maternal or foetal hyperglycaemia provided the blood traverses the placenta. That this is true is shown experimentally, for when a foetus is made hyperglycaemic and cut off from its placenta, by clamping the umbilical vessels, fructose is not produced, whereas if the foetus is removed from a hyperglycaemic ewe but the placental circulation is maintained by means of a pump, then fructose appears in the placental circulation. Other experiments show that the foetal blood glucose is the source of the fructose. The total reducing substance in the foetus may be greater than that of its dam because of the formation of fructose from glucose by the placenta.

Since the author's experiments show that the passage of glucose across the placenta to the foetus is quick and results in some fructose formation, and that in the case of fructose introduced into the maternal blood passage to the foetus is slow, they suggest (in agreement with other investigators) that the placental passage of these two sugars is a more complex process than simple diffusion. Furthermore, glucose will pass across the placenta in both directions, but fructose only from dam to foetus.

F. R. B.

* **The Origin of the Blood Fructose of the Foetal Sheep.** Huggett, A. (1951.) *J. Physiol.* **113.** 258–275.

* * * * *

Fermentation in the Rumen of the Sheep*

III.—Five grammes of ground wheaten hay chaff were incubated in 150 ml. rumen liquor, enriched with ammonium carbonate under an atmosphere of carbon dioxide. The sheep used as a source of rumen liquor was fed on wheaten hay chaff. The fermentation was followed up to 48 hours. Sixty per cent. of the total fatty acid production occurred during the first 12 hours ; propionic acid was formed in greater quantity during the first four and a half hours but thereafter more acetic acid than propionic acid was produced. At all times, however, proportionally less acetic acid and more propionic acid was present than in the initial rumen liquor.

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IV.—Carboxyl-labelled (C^{14}) acetic or propionic acids were introduced into the *in vitro* fermentations of wheat (six experiments) and lucerne hay (one experiment) using rumen liquor as inoculum. The acids formed were separated chromatographically and the activity of each acid measured. At the end of the fermentation period (48 hours) carboxyl carbon from acetic acid was found in propionic, butyric, valeric and in higher acids ; in most experiments the greatest count besides that of acetic acid itself was found in butyric acid. With labelled propionic acid little or no activity was found in acetic acid and none in butyric acid : considerable activity appeared in valeric acid, however, and small counts in capric and higher acids.

Analysis of rumen liquor showed that in addition to the well-recognised acids small quantities of formic acid, *iso*-butyric acid (3 to 8 per cent. of the total butyric fraction), *n*-valeric acid and an unidentified isomer, capric acid, and

(*Concluded at foot of column* 1, *page* 552)

* **Fermentation in the Rumen of the Sheep.** III.—Intermediate Stages in the Fermentation of Wheat or Hay *in vitro* by Micro-organisms from the Rumen ; IV.—The Nature and Origin of the Volatile Fatty Acids in the Rumen of the Sheep. Gray, F. V., *et al.* (1952.) *J. Exp. Biol.* **29.** 54–56 ; 57–65.

REPORT

[**Non-Pulmonary Tuberculosis of Bovine Origin in Great Britain.** By G. S. Wilson, J. W. S. Blacklock and Lilian V. Reilly. (1952.) Pp. 108. National Association for the Prevention of Tuberculosis, Tavistock Square, London, W.C.1. Price 16s.]

As a result of recent legislation which enforces the pasteurisation of milk in the large urban areas and of the progress of the Attested Herds Scheme, there is a tendency to complacency in regard to the dangers of the bovine type of organism in cases of human tuberculosis. The report published under the auspices of the National Association for the Prevention of Tuberculosis is particularly timely because it brings into sharp relief the hazards to which children and young adolescents are exposed.

The report is divided into three parts. In parts 1 and 2, which have been drawn up by G. S. Wilson and J. W. S. Blacklock, the incidence of non-pulmonary tuberculosis in England and Wales is discussed. In a table on page 5 it is shown that in England (1943-1945) the percentage of bovine infection in meningitis cases was 28 of the total, and in surgical cases 25·7. The corresponding figures in Wales (1943-1945) were 10·1 per cent. in meningitis and 27·9 per cent. in surgical cases. In Scotland (1943-1944) the percentage of meningitis cases was 11·1 and surgical cases 33·8. It is pointed out that an appreciable proportion of milk given to children in schools, especially in small towns and country districts, is not rendered safe. It is claimed that in England about 7 per cent. of milking herds are supplying raw milk containing tubercle bacilli: as a result children attending school are now more likely to be affected than pre-school children. Earlier figures supplied by Griffith showed that bovine infections were considerably higher in the nil to four years than in the 5 to 14 years age group, whereas in the present series the proportion was about the same with the highest incidence in the five to nine years age group. Many of the records suggest that the consumption of raw milk must have been only slight or occasional, pointing to the danger of drinking raw milk at any time or in any form.

In Wales the proportion of the infections with the bovine type was only 17 per cent. compared with 26·3 per cent. in England. Some attempt is made to assess the effect of the Attested Herds Scheme, and it is suggested that the fact that the proportion of bovine infections is lower in Wales than in either England or Scotland may be associated with the higher number of attested herds in Wales in relation to the total cattle population.

The detailed results obtained by the survey in England and Wales are summarised in a series of tables indicating distribution of patients according to geographical regions, age, sex, contact history, calculated number of deaths from non-pulmonary tuberculosis of bovine origin, and so on. Susceptibility does not seem to be confined to children: approximately 30 per cent. of the cases occurred in persons over 15 years of age. It is suggested that immunity to bovine infection is not a natural concomitant of adult life.

In Scotland during the war period an increase in tuberculosis occurred, the number of deaths rising rapidly to the maximum in 1941, and thereafter tending to fall. In general the proportion of bovine cases of meningitis is about three times as high in the country as in the town, and in surgical cases, twice as high. Of the 560 strains from meningeal cases 11·1 per cent. were bovine, and 33·8 per cent. of the 491 from surgical cases. Nearly 80 per cent. of the cases infected with bovine bacilli habitually consumed raw milk as against 46·5 per cent. of those infected with human strains. A higher proportion of cases from rural areas were affected with bovine bacilli.

Part 3 covers the investigation in Northern Ireland by Lilian Reilly. The results seem to show that there has been a marked decline in the incidence of bovine tuberculosis during the past 10 years, coinciding with a great increase in the pasteurisation of milk, widespread tuberculin testing of cattle, and an increased awareness by the public of the risks of drinking milk from tuberculous cattle. It seems incontrovertible that the decrease in tuberculosis due to the bovine type in Northern Ireland must be due to the increased amount of pasteurisation being carried out. Only one case of bovine-type pulmonary tuberculosis was found out of more than 1,000 cases: the proportion of bovine cases in the non-respiratory group was 4·3 per cent. R.E.G.

(*Continued from page 551*)

traces of a higher acid were present. The composition for sheep fed wheaten or lucerne hay is summarised as follows:

	% of total
Formic acid	0–5
Acetic acid	62–70
Propionic acid	16–27
n-butyric acid	6–11
iso-butyric acid	0·3–0·6
Valeric acid*	1·6–3·2
Capric acid	0·5–1·0
" Heptoic acid*"	0·04–0·05

* Including an unidentified isomer.

A. T. P.

NOTES AND NEWS

Diary of Events

Sept. 19th and 20th.—Meeting of The Biochemical Society, in the Department of Biochemistry, The University, Glasgow. 11 a.m. both days.

Sept. 22nd.—Annual General Meeting of the Veterinary Section of the Universities Federation for Animal Welfare, at Westham House, Barford, near Warwick, 2 p.m. (See Notice.)

Sept. 24th.—Meeting of the R.A.V.C. Division of the B.V.A. at the Equine Research Station of the Animal Health Trust, Newmarket, 11.15 a.m.

Oct. 4th.—Autumn Meeting of the North of Scotland Division, B.V.A., at Inverness (Drumossie Hotel), 4.30 p.m.

Oct. 7th.—Meeting of the Lincolnshire and District Division, B.V.A., at the Grand Hotel, Peterborough, 2.15 p.m.

Oct. 8th.—Meeting of the Editorial Committee, B.V.A., at 36, Gordon Square, W.C.1, 2 p.m.; Executive Committee to follow immediately. N.B. *There will be no meeting of either the Editorial Committee or its Executive Sub-committee in September.*

Oct. 23rd.—Annual Dinner and Dance of the Midland Counties Division, B.V.A., at Birmingham (Midland Hotel): Reception, 7.30 p.m.

THE B.V.A. AT HARROGATE

This year the members and delegates, their ladies and other visitors met at Harrogate for the 70th Annual General Meeting and Congress of the British Veterinary Association. The distinction of acting as host Division to the first conference to be held by the parent body under that new and handy name has fallen to the Yorkshire Veterinary Medical Society, with the appropriateness due, historically, to a long and honourable tradition as a strong and influential corporate unit and, immediately, to the fortunate availability of its able and energetic Hon. Secretary, Mr. K. G. Towers, of the Department of Agriculture in the University of Leeds, to undertake the onerous duties of Local Hon. Secretary of the Congress, with that experienced stalwart on the spot, Mr. J. O. Powley, as Chairman of the Provisional Committee. Past triumphs in the county also add their weight, and many have happy recollections of the fine gatherings at York and Scarborough. Indeed, there may well be some present with us this week, from September 7th to 13th, who recall as participants the much earlier event at Harrogate itself: the 27th two-day General Meeting of what was then known as the National Veterinary Association, held at the George Hotel in July, 1909. Nowadays a full week scarcely suffices to cater for the social and instructional requirements of a membership which, in the interval, has grown from 365 to a figure within sight of 6,000. Yet it is not only in the trencherman sense that they had good fare in those days, for all present at "The George" would regard as in itself well worth the journey an opening paper on lameness, by Professor Macqueen, and a discussion occupying four hours and 80 pages of the *Proceedings*. To-day that modest company is multiplied many times, while in place of the subjects discussed at the hostelry (now awaiting derequisition !), we have, *inter alia*, electrotherapy and electro-cardiography, the domestic animals as a source of infection to man, and modern insecticides and parasite control, although another equine topic of that far-off meeting, "Our Horse Supply," certainly would have borne re-introduction!

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On entering Harrogate, the phrase comes to mind "not only pre-eminent as a spa, it is also a holiday resort of distinction." Pure "guide book," but in the true tradition of the place and deriving from those Edwardian days when the elegant horse-carriage was the vogue and to the world of fashion "The Stray" an agreeable alternative to Rotten Row or the gracious sidewalks of Bath or Tunbridge Wells. The attraction of the town, or its lovely countryside of moors and dales, drew many of our present company there for the official opening on Monday last. For these it was a great pleasure to be the guests of the "Yorkshire" at the eve of Congress "At Home" and to enjoy both their whole-hearted welcome, voiced by their President, Mr. H. B. Allan, and their traditional hospitality at this Sunday function at the Hotel Majestic—the Congress Headquarters.

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The opening, foregathering, hour of Congress, in common with Harrogate itself, possesses "an air" of its own, compounded of vivacity, expectancy and communal good humour. The requisite infusion of decorum is soon forthcoming, however, in the dignified opening ceremony. This duly took place in the impressive conference chamber—The Royal Hall—when the Association was accorded a civic welcome by the Mayor, Councillor A. V. Milton, J.P., whose spontaneous cordiality was reciprocated in kind and in full measure by our President, Mr. A. J. Wright, of Macclesfield, who also thanked the Mayor and Council for the admirable facilities so readily afforded. The President also welcomed the large number of delegates and guests from overseas, from the various government departments, local societies and representatives of students and other bodies. A move was then made to the Royal Hall Annexe, where was housed the admirably staged and impressive display of drugs, instruments and appliances, which the President commended to the attention of the members, after congratulating the exhibitors on opening to time in exceptionally difficult circumstances.

In line with the procedure at Eastbourne last year, as a prelude to the discussion of the previously distributed papers, the opening afternoon was devoted to an address of somewhat wider application, by one of international repute. On this occasion the guest speaker was Dr. K. V. L. Kesteven, an Australian graduate of Sydney University Veterinary School, Chief of the Animal Production Branch of the Food and Agriculture Organisation, who gave a masterly and stimulating review of its place in the United Nations system, and its activities in relation to animal husbandry, range and pasture management, breeding, and the control of diseases of livestock, notably rinderpest, foot-and-mouth disease, and Newcastle disease.

An opening day, full of interest, was brought to a memorably enjoyable close by the civic reception and dance given by the Mayor and Mayoress in the Corporation Lounge Hall, beautifully decorated for the event. Here the manifestly sincere welcome accorded the members was renewed, their appreciation being intensified thereby and through the most hospitable entertainment provided.

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The proceedings of Tuesday largely exemplified the extent to which the scientific programme broke modern ground. At a finely attended morning session Mr. T. N. Gold, of Redditch, dealt with the technical and practical aspects of the application of electro-therapy in the horse, stressing the advance on "line firing" represented by short-wave inductotherapy, with controlled exercise, for the treatment of strains of ligaments and muscles, or of lacerations. Dr. A. C. Fraser, of Aldbourne, was the opener of a brisk discussion. Another good attendance greeted Mr. W. C. Miller and Colonel J. Hickman, of the Animal Health Trust's Equine Research Station, when in the afternoon, with the aid of lantern slides, they elaborated their thesis on the subject of electro-cardiography in the horse, describing the types of apparatus employed and the uses and interpretation of electro-cardiograms, together with a provisional assessment of the potential practical value of the resultant records. The latter paper was preceded by a helpful talk and discussion (for B.V.A. members only) on "Diagnosis of Foot-and-Mouth Disease in the Field," the address being given by the incoming President, Mr. A. M. Graham, of the Ministry of Agriculture.

Meanwhile, the Harrogate hinterland being a realm of genuine reward for the excursionist, a large group representative of that diminishing number of ladies who have no direct interest in our scientific sessions visited lovely Fountains Abbey and also Ripon, claimed to be the second oldest city in the country, which takes a great and justifiable pride in its cathedral. In the evening all joined in what after all is the social hub of the Association's life—the President's reception and dance, where, at the Hotel Majestic, Mr. and Mrs. Wright entertained a great company whose presence was of the nature of homage to a highly esteemed and most popular President and lady.

(*To be continued.*)

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THE ROYAL ARMY VETERINARY CORPS OFFICERS' CLUB

A glance at the Register of Veterinary Surgeons will reveal that a very high proportion of the members of the Royal College have served in the armed forces and of these the great majority have held commissioned rank in the R.A.V.C. This is a record of which we can be proud. It is the custom in the various units of the service for the officers, serving and retired, to join together and form an association or club. Such a club enables them to meet together occasionally so that old friendships made in peace or war, at home or abroad, may be renewed and new contacts made; also that matters which have to do with the interests of the Corps may be brought to notice, discussed and if need be implemented by action.

The Club, too, has a benevolent side, so that assistance may be given in time of need to those officers or their dependants whom good fortune has passed by.

The birth of the R.A.V.C. Club may be said to have occurred back in 1880, for that was the date of the first annual dinner of the Army Veterinary Department. In the year 1920 and at the instigation of Major-General Sir Layton J. Blenkinsop, this association of officers, who met over the festive table, was extended to form the Club which we know to-day. The Club has been a success socially and it has done great work in the field of benevolence. It has done much to further the interests of the Corps, and knit the members together in a happy association.

At the recent annual general meeting the feeling was expressed that the membership of the Club is not as large as it might be. It was thought that this may be due to the fact that many ex-officers are not fully aware that such a club exists or what its functions are.

It was resolved that through the courtesy of the Veterinary Press the club should be brought to the notice of all ex-officers of the Corps and that they be cordially invited to become members. The subscription is 10s. 6d. per year.

The annual reunion dinner is the great event of the year, but there are various other activities of a sporting and professional nature while, as indicated above, the Club undertakes charitable work for the relief of distress amongst ex-officers of the Corps and their families.

Lieut.-Colonel J. Clabby, the War Office (V. & R.), Droitwich, Worcs, is the Hon. Secretary and he will be pleased to send full particulars of the Club and its activities to anyone who may be interested.

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PERSONAL

London Ministry Post for Mr. John Reid

Mr. John Reid, M.R.C.V.S., D.V.S.M., Divisional Veterinary Officer for Cumberland under the Ministry's Animal Health Division since 1944, is moving to London and a new post as Superintending Veterinary Officer stationed at the Ministry's Head Office.

A native of West Perthshire, Mr. Reid graduated from Edinburgh in 1929. He first went to the Carlisle area in 1932 when appointed Assistant Veterinary Officer to Cumberland County Council and was stationed in Carlisle until 1938 when the veterinary services in the country were taken over under the reconstituted Animal Health Division of the Ministry.

In 1938, on joining the Ministry, he became Divisional Veterinary Officer for the County of Brecon in South Wales; in 1942 he was seconded to the Head Office of the Ministry in London and in 1944 he returned to Cumberland.

Mr. Reid is succeeded in Cumberland by Mr. J. K. S. Elmslie, M.R.C.V.S., of the Ministry's veterinary staff.

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Births.—Lucas.—On August 25th, 1952, at Princess Elizabeth Hospital, Nairobi, Kenya, to Dr. Shelagh (*née* Fair), wife of J. M. S. Lucas, M.R.C.V.S., Box 2141, Nairobi, a daughter.

Milnes.—On September 2nd, 1952, to Deirdre M. Milnes (*née* Kenny), M.R.C.V.S., and John N. Milnes, M.D., of 61, Laurel Way, Totteridge, N.20, a sister (Clare) for Anthony and Pauline.

Muir.—On August 26th, 1952, to Denise (Rowe) and Thomas Howie Muir, M.R.C.V.S., Blank House, Wymondham, a second daughter—Denise Valerie.

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Marriage.—Snowden—Thorne.—On August 9th, 1952, at Llandaff Cathedral, Cardiff, James Stanley Snowden, M.R.C.V.S., of Exeter, to Anne J. Thorne, B.A., second daughter of Mrs. Annie and the late Arthur Thorne, of Cardiff.

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Will.—Mr. Joseph Blake Walker, Monks Pond Cottage, Binfield, Berks, veterinary surgeon, left £31,466 (£31,735 gross).

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R.C.V.S. OBITUARY

Hunter, Thomas Stott, 98, Grove Lane, Camberwell, S.E.5. Graduated London, December 15th, 1903. Died August 30th, 1952; aged 69 years.

Stainton, Frederick Harold, 7, Bark Place, W.C.2, and formerly of 167, Church Street, Kensington. Graduated London, July 9th, 1914; Fellowship February 19th, 1921. Died September 8th, 1952.

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Mr. Frederick Harold Stainton, F.R.C.V.S.

As we go to press we learn with deep regret of the death of Mr. Frederick Harold Stainton. Mr. Stainton was a distinguished West London practitioner, especially well known in the dog breeding and showing world; he was responsible for the veterinary inspection arrangements at all the major London shows. Further reference will be made in an early issue.

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FOOT-AND-MOUTH DISEASE CONFERENCE AT LYONS

Dr. J. A. Galloway, Director of the Research Institute at Pirbright, was among those invited to attend a conference recently held at Lyons under the auspices of the Ministry of Agriculture and the Institut Français de la Fièvre Aphteuse. Those participating were practising veterinary surgeons and officials of the French Ministry and its purpose was to exchange information about the epidemic of foot-and-mouth disease which raged this summer in France, and about the methods which have been developed to combat it.

It had been announced previously that a new method of virus culture should make it possible to increase 300-fold the quantity of vaccine which can be produced from one sick animal. The method was discovered at the veterinary research centre of Maisons Alfort, near Paris, by Professor André Thomas and a team of research workers.

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax:

Berks.—Avington Farm, Hungerford (August 29th); Tidcombe Cottage, Kintbury, Newbury (September 4th).

Beds.—Clifton Bury Farm, Clifton, Shefford (September 5th).

Cambs.—Wyndmere, Steeple Morden, Royston (August 30th); The Homestead, 186, High Street, Cottenham (September 1st).

Ches.—Brook Villa, Bulkeley, Malpas; The Kennels, Park View, Combermere, Nantwich (August 30th).

Devonshire.—Kingsleigh Farm, Churchinford, Chard; Hillside, Crookmans Corner, Goodleigh Road, Barnstaple (August 29th); Bingwell Farm, Tiverton (September 4th).

Glos.—Cocklebarrow Farm, Aldsworth, Cheltenham (August 30th).

Hunts.—Grange Farm, Houghton (August 29th).

Kent.—2, Crow Hill, Downe, Farnborough (August 30th); Rose Cottage, Westwell Leacon, Ashford; Colinett Farm, Brasted, Sevenoaks (September 1st); Orwell, Blackness Lane, Keston (September 3rd).

Norfolk.—Old Hall Farm, Colton, Norwich; Dairy Farm, Marsh Road, Halvergate, Norwich (August 29th); Park Farm, Horstead, Norwich (September 3rd); Lyng Easthaugh, Norwich, Norfolk (September 5th).

Oxfordshire.—Manor Farm, Toot Baldon, Oxford (September 2nd); High Coggs Farm, Witney (September 4th).

Somersetshire.—The Hammocks, Charity Farm, Upton, Long Sutton, Taunton; Knaphill Farm, Bath Road, Wells (August 29th); Beech Tree Farm, Upton Noble, Shepton Mallett (August 30th); Charity Farm, Lovington, Castle Cary, Bath; Walronds Park, The Brewers, Taunton (September 1st); Home Farm, Compton Pauncefoot, Yeovil; Ashway Farm, Dulverton (September 2nd); Stalls Farm, Upton Noble, Shepton Mallet (September 5th).

Suffolk (E.).—Charters Farm, Worlingworth, Woodbridge (August 29th).

Suffolk (W.).—Gate Farm, Lawshall, Bury St. Edmunds (September 2nd); The Goodlands, 55, Swan Street, Boxford, Colchester (September 3rd).

Wilts.—Uffcott Farm, Broad Hinton, Swindon (September 1st); Littlecott, Park Farm, Ramsbury, Marlborough; Lypgate Farm, Kington Langley, Chippenham (September 3rd); Smallholding, London Road, Shrewton (September 5th).

Yorks (W.R.).—Acton Flatts Farm, Lindley Moor, Flixby, Huddersfield (September 1st); Manor House Farm, Rimington, Clitheroe (September 3rd).

Foot-and-Mouth Disease:

Kent.—Langdon Farm, Ruckinge, Ashford (September 5th).

Sussex (W.).—Polson Dairy Farm, Lidsey, Bognor Regis (September 4th); Beeding Court Farm, Upper Beeding, Steyning (September 5th).

Fowl Pest:

Essex.—Linley Hall, Point Clear, St. Osyth, Clacton-on-Sea (August 29th).

Isle of Ely.—Slaves Hill, Doddington, March (August 29th).

Swine Fever:

Beds.—The Elms, High Street, Arlesey (September 4th).

Ches.—Pool Farm, Heath Road, Runcorn (September 1st).

Cornwall.—The Old Cottage, Holywell, Cubert (September 4th).

Derbyshire.—Highfield Allotments, Highfield Road, Chesterfield (September 3rd); The Nurseries, East Street, Scarcliffe, Chesterfield (September 4th).

Durham.—Cemetery Road Piggeries, Southwick, Sunderland (September 4th).

Lancs.—Summerfield, White Lund, Morecambe (September 5th).

Lincs (Holland).—Mayfield, Willington Road, Kirton End, Boston (September 1st); Main Road, Butterwick, Boston (September 2nd).

Staffs.—Woodland Lane, Four Crosses, Hatherton, Nr. Cannock (September 2nd).

Warwicks.—Broomhill Farm, Stratton on Dunsmore, Rugby (September 1st); South Road Piggeries, Northfields, Birmingham (September 3rd); Lodge Green Farm, Meriden, Coventry (September 5th).

Worcs.—Cherry Side, and Blue Ball Farm, Far Forrest, Kidderminster (September 2nd).

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LEGAL NOTES

Failure to Notify Foot-and-Mouth Disease

Charged with failing to give notice with all practicable speed of the existence of foot-and-mouth disease on his premises, a Cumberland farmer, Henry Edward Hedley, Denton, Guilsland, appeared before the Brampton magistrates on August 27th and was fined £30 with 3 guineas costs.

A Ministry of Agriculture veterinary inspector said that when he visited Hedley's farm he found nine out of 15 pigs affected by the disease. Making his routine examination he found a Shorthorn cow which, he said, had been affected for at least five days. Hedley, when he notified the Ministry Centre at Carlisle, only mentioned the pigs.

Hedley said he knew now that the pigs had the disease from milk fed to them from diseased cattle. He had not noticed anything wrong with the Shorthorn cow.

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Two Fined for Operation on Dog.—In our last issue we reported that the Southampton magistrates had issued a warrant for the arrest of Thomas McGowan, of Southampton, who failed to appear on charges of operating on a dog without using sufficient anaesthetic, and of practising veterinary surgery without being registered. Charged with McGowan was William Cole, of the same town. When the charges were heard on August 29th, each was fined £5 on the first and £2 on the second, and each had to pay 3 guineas costs.

McGowan, giving an address at Hemel Hempstead, pleaded guilty to the first charge and not guilty to the second. Cole pleaded not guilty on both counts.

Mr. Alan Woodford prosecuted for the Royal College of Veterinary Surgeons.

Inspector William Hallam, of the R.S.P.C.A., said he asked McGowan about the operation. He replied: "Why not? I have every right. I have done hundreds of pigs and sheep in Ireland. I have got a certificate to do it."

McGowan said the certificate was issued to operate on animals on a farm.

Inspector Hallam said Cole told him: "I thought it was all right as McGowan said he held a certificate to do this sort of thing in Ireland. I did not know I was doing anything wrong."

It was stated that a dirty penknife and razor blades were used to operate on a terrier dog in a filthy cellar in Kingsfield Road, Southampton, and that no anaesthetic was given.

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R.S.I. CONGRESS AT HASTINGS, 1953

Lord Eustace Percy will be President of the Health Congress which is being organised by the Royal Sanitary Institute at Hastings from April 28th to May 1st, 1953. He is a former President of the Board of Education and was for many years Member of Parliament for Hastings.

It is expected that over 2,000 delegates will attend the Health Congress from all parts of the world. Papers will be presented by medical officers and doctors, veterinary surgeons, engineers, architects, surveyors, sanitary inspectors, chemists, health visitors and others, a number of the contributors coming from overseas. The delegates will also visit places of public health interest in Sussex.

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ELECTRIC WHALING

Under the above heading, Major C. W. Hume, Director of the Universities Federation for Animal Welfare, contributes to a recent issue of *The Times* the following letter on a subject that has been dealt with with in our own columns from time to time:—

"On January 3rd you published a brief review of the progress in electric whaling, indicating that while it was then possible to adapt the old-fashioned Svend-Foyn gun and heavy harpoon for the purpose of electrocuting whales, a light harpoon with much better ballistic properties was being developed; it is a hollow harpoon fired from a spigot. As you will be aware, the prototype of this weapon has just received its final sea trials and been pronounced satisfactory. In view of its technical advantages there can be little doubt that it will eventually displace the traditional equipment, though the manufacturers may not be able to fit our more than, say, half a dozen catchers this year. The flatness of the trajectory should give a much more accurate aim. The instant immobilisation of the whale will obviate the expenditure of half, one, or more hours in tiring the animal out, and with the abolition of the explosive head none of

the meat will be rendered inedible by laceration or incipient putrefaction. There will be a saving of cost in lost harpoons and whales and in expenditure of whale ropes and forerunners. The saving of time should make it possible to shorten the season at the earlier end and so allow the whales longer than at present for fattening.

"Above all, the horrible cruelty of the traditional method will be got rid of. It is not at present possible to say which of the three main causes of death by electrocution is usually responsible for the death of the whale, but even if the killing is not absolutely instantaneous it will be an incomparably briefer process than the long-drawn-out horrors of the old system. When a fine deed has been done it ought to be put on record for the sake of example. Sir Vyvyan Board, a veteran of the first world war, went to the Antarctic in 1949-50 and himself served the gun in the bow of a catcher, sometimes for 15 hours a day, under conditions that most younger men would not be tough enough to stand. His harpoon burst more than once. Except for Mr. Robert Marsden of the General Electric Company, who accompanied him and also deserves our gratitude, everybody else in the Antarctic at that time assumed that electric whaling was a freak idea to be laughed out of court. Nobody except Sir Vyvyan Board could have seen the thing through; by the mercy of Providence he happened to be both inventive and humane, and did it."

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BLOWFLIES AND MEAT

In its last Pest Infestation Report the D.S.I.R. have the following comments to make upon the blowfly:—

"Work on blowflies in slaughterhouses has been continued to find out what the severity of the 'blown' carcase problem is, the species of fly causing the damage, the most attractive kinds of meat, and so on. It has been found that bluebottles, although they are only 5 per cent. of the population, cause more than 90 per cent. of the damage.

"Extensive trials of methods of control at breeding places have been completed. Recommendations can now be made for reducing infestation at refuse depots, etc., to 1 per cent. or less of its original level.

"A large multiple store collaborated in experiments on the overwintering of flies. Seven premises were searched during the winter. Only in one store were a few flies found. It seems unlikely that they stay in the stores and reinfest them in the following spring. Thus the problem of how flies spend the winter remains unsolved."—*Meat Trades Journal.*

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SWINE FEVER

The outbreaks of swine fever confirmed from January to the end of August reached a total of 627—a figure slightly higher than for the same period of 1951. Progress of the disease this year, however, has been steady and has not been marked by the steep rise in July and August which took place in 1951. Areas chiefly affected are Lancashire, Warwickshire, Staffordshire, Essex, Surrey, Northamptonshire, Derbyshire and Hertfordshire.

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The Ministry of Agriculture and Fisheries announces that the price paid for "Gay Time" recently purchased by the National Stud from Mrs. J. V. Rank was £50,000.

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ANTHRAX

A disturbing feature of the "Disease of Animals Returns" from the Ministry during recent weeks has been the sharp increase in the number of outbreaks of anthrax. Already this year there have been about twice the number of cases reported in the whole of 1949, in which 244 cases were confirmed; in 1950, there were 344 and last year 407.

From August 16th to 31st, no fewer than 71 cases were confirmed, bringing the total for the year, to the end of the month, to 487. The number now being reported averages from six to seven daily, spread over many counties.

Increases in the numbers of outbreaks in the past three and a half years follow a long period during which outbreaks were getting progressively fewer.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

TRAINING IN TROPICAL VETERINARY MEDICINE

Sir,—The editorial of July 26th on "Veterinary Parasitology and Tropical Medicine" and the correspondence which has followed could not have appeared at a more opportune time, as they add weight to the efforts which this School is making to meet the growing demands from overseas countries for training in Tropical Veterinary Medicine.

Since 1930, through the co-operation of the Royal (Dick) Veterinary College with the University of Edinburgh, a course of study leading to the Diploma in Tropical Veterinary Medicine (D.T.V.M.) of the University of Edinburgh was a joint responsibility, but since the outbreak of war in 1939 the course and examinations for the Diploma have been in abeyance.

Shortly after the R.C.V.S. Council, over two years ago, discussed the necessity for Tropical Veterinary Medicine courses, a joint committee of teachers, representing departments of the University of Edinburgh and the Royal (Dick) Veterinary College primarily interested in Tropical Veterinary Medicine training, held a number of meetings to consider the revival of the course in Edinburgh. The original curriculum was revised in the light of previous teaching experience, but before finally drawing up its report the Committee decided to get the views of Senior Colonial Veterinary Officers and others interested in veterinary education on the proposed curriculum of study, and for this purpose Dr. A. Robertson was sent on a tour round Africa during the summer of 1951, to consult them and gain first-hand knowledge of their problems and difficulties. Following Dr. Robertson's report on his visit to Africa, the regulations and curriculum were amended and the Committee's report submitted through the Faculty of Medicine was approved by the Senatus, who recommended that the course and Diploma in Tropical Veterinary Medicine should be resumed in the Session 1952-53. Unfortunately it will not be possible to start classes during this coming Session, as certain financial requirements at present before the University Grants Committee have not yet been settled.—Yours faithfully, W. M. MITCHELL, Director of Veterinary Education, Royal (Dick) School of Veterinary Studies, University of Edinburgh. *September 2nd*, 1952.

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THE DESIGNATION "GELDING"

Sir,—I was more than interested in Mr. Tutt's letter in the issue of September 6th, 1952.

I do not think any colleague would interpret a gelding as other than a male equine whose testicles have been removed.

Naturally and in practice sexual desire disappears together with the subsidiary symptoms attendant on the presence of testicular tissue.

I have more than an ordinary interest in the case which prompted Mr. Tutt's letter, being asked, in the first instance, to examine the "gelding" when visiting a client's farm where he was temporarily kept. The history given was that in the presence of other horses he behaved like a "rig" and that he was continually nibbling and catching hold of the owner, a young lady, and would eject and erect his penis, which by the way was well developed. He was examined by me in a loose-box at the farm buildings where no horses are kept and the owner handled him. He behaved exactly as complained of and on close examination I found the stump of a cord on the left side. Being satisfied some testicular tissue was present, I gave it as my opinion to the owner and her parents that he was not a gelding and from his behaviour suggested it was not prudent for the young lady to handle him. It transpires that following representations made to the seller it was agreed that Mr. Tutt should act as referee and if in his opinion the horse was not a gelding it would be returned.

Even though Mr. Tutt has informed me that "it is well known that some geldings do behave like a rig in spite of castration," my own opinion is, and experience has taught me, that when the symptoms described are exhibited then the animal is either a "rig" or is "rough cut" and at the minimum indicates the presence somewhere of testicular tissue.

Such a case does not come under the category of "gelding."—Yours faithfully, G. WARNOCK BEGG, The Lower House, Wickham, Fareham, Hants. *September 5th*, 1952.